however, be fatal, and are usually followed by an aggravation of existing mental and bodily symptoms.

The mental symptoms of the fully developed stage are a continuation of those of the later prodromal period. Usually before the physical signs of the developed disease have become well marked, the extravagances of conduct and of advanced dementia and evident delusions have made the insanity manifest to friends and the public. In fact, the mental often precede the physical symptoms. Some patients pass gradually into an active delusional insanity with comparatively slight physical symptoms, others have some of the bodily symptoms very pronounced with apparently little intellectual impairment. Most patients, however, after the speech defects have become permanent or after a period of months, or it may be of years, of the condition described as the preliminary stage, begin to exhibit an intensification of their mental symptoms. The tendency toward dementia becomes evident, and a peculiar delusional type is often developed, there are grandiose ideas of wealth, personal ability, etc. These may have existed before to some extent, but they now become conspicuous, and in the beginnings of this stage the mental state may closely resemble that of acute simple mania. With the abnormal excitation of the faculties, however, there is a more pronounced element of mental weakness and confusion than is the case with the acute maniac. Later, as the bodily symptoms develop, the dementia is more prominent, the mental activities take on the delusional form, and the extravagance of the patient's fancies is uncontrolled by any element of reason or recognition of possibilities or probabilities. It is not usual in this stage to see fixed and consistent paranoic conceptions, though in some instances such occur. In the majority of this type of paretics the delusions are unsystematized—they are the expression of the exalted self-consciousness of the individual and the advancing incapacity to recognize the true relation of things. These patients are possessors of billions of dollars, have mines where coined double eagles are dug out by the ton, are husbands of thousands of wives, own all the railroads, are the greatest orators and authors, have converted millions by their sermons, etc., all given out as gospel truth in rapid succession it may be by the same individual. This unsystematized and extravagant character of paretic delusions is a feature of the disease in so large a proportion of the cases that it has been held and is still often considered to be the special characteristic of the disease. Not less frequently, however, at least of late years, other types of mental disturbance are observed. In a certain proportion of cases this is of a depressed type throughout, and often a hypochondriacal extension of the neurasthenic depression of the earliest phase. These patients may show a mental weakness only and a general tendency to exaggerate and invent morbid feelings; any slight ailment or injury disturbs them unduly and is sometimes the direct occasion of an outbreak of delirium. In other cases there are the self-accusatory delusions and feelings of unworthiness, as in melancholia, which is strongly suggested; and in still others only a progressive dementia from the beginning to the end of the disease. Occasionally paresis assumes a circular type of alternating excitement and depression, and there are cases in which fixed delusions resembling those of systematized delusional paranoia occur and continue throughout a considerable period. I have seen a paretic who for five years remained in a stationary condition with fixed moderately exalted delusions, pronounced visual hallucinations of angels, deities, etc., a rather prominent general dementia, speech disturbances marked, pupillary inequality, and a generally good physical state as regards nutrition, the digestive functions, etc. Then he rapidly passed into the third stage, became paralytic, and soon succumbed.

It is difficult to estimate the proportion of paretics in which the disease assumes the exalted and the depressed or demented types; but while the former is usually considered the most typical, it is certainly not the most frequent. The demented type, in which the mental symptoms are predominantly those of steadily progressive mental failure, is perhaps as frequent as any; Kraepelin estimates that it includes over forty per cent. of the cases according to his experience, while the exalted form includes only fifteen or sixteen per cent., or, taking in the more acutely maniacal cases which he classes as the agitated type, about twenty-six or twenty-seven per cent. The depressive type he finds to occur in about twenty-seven per cent. of all cases. The purely hypochondriacal and the circular cases and those with only comparatively slight intellectual disturbance even with marked physical symptoms form a still smaller contingent, but they exist as a class and may be reckoned according to the writer's observation as forming two or three per cent. of the whole. The difficulty in any classification is the large number of cases in which the symptoms are at one time of one type and at another show a quite different aspect, so that it is not easy to say in just which class they belong. In women these irregular forms are proportionately more frequently observed than in men, but the number of female paretics observed by me has been so small that a positive generalization is hardly justifiable.

Sensory symptoms may occur at all stages of the disease after it has once developed. Anæsthesia and analgesia are not uncommon in advanced cases, and various paræsthesias may occur in the earlier stages. Disorders of special senses are common, especially of smell or taste; depraved appetites are often observed. The sexual appetite is sometimes exalted in the excited cases, seldom so in the depressed, and rarely if ever in the progressive dementia type (Marandon de Montyel).

Hallucinations are not so prominent in paresis as in some other forms of insanity, but they occur in a considerable proportion of all cases. In the agitated type of either expansive or depressive delusion they are apt to be manifest. In the agitated depressed cases visceral hallucinations are comparatively common, the patients complaining of having cats, dogs, reptiles, etc., inside them, and in women delusions of pregnancy are aroused. Sometimes visual and auditory hallucinations are also common, but are not so strikingly frequent, except in the delirious attacks.

A not very uncommon feature in the fully developed stage and sometimes beginning it are the occasional attacks of acute maniacal excitement, usually of short duration, but sometimes prolonged over days and even weeks. They may occur suddenly in the night or in the day, and suggest the psychic equivalents of the epileptic. It is quite possible that the mechanism is the same, that they are the equivalents in a sense of the apoplectiform or convulsive attacks, symptoms of special cortical explosions, only that here the objective manifestation is psychic in its character. Like the convulsions or congestive attacks, they are likely to be followed by a usually permanent psychic deterioration,—they accelerate the progress of the dementia.

Of quite a different character are the attacks of acute delirium that occasionally occur at any stage of the disorder. They are sometimes incited by apparently a trivial cause, such as a slight traumatism in the hypochondriacal cases, but they form one of the most formidable complications when they occur. The patient passes into a wild maniacal delirium, often with hallucinations, his bodily activity is incessant and violent, food is refused, insomnia is nearly absolute, sleep being only in snatches from exhaustion, the bodily temperature rises sometimes to an extraordinary height—in a case observed by the writer it was 107° F. in the axilla through the shirt several days prior to death. Toward the close of the scene it falls again as the vital powers fail and the patients succumb to exhaustion, and the rectal temperature may even be below normal some time before death. In some cases, in which the symptoms are less severe, the patient may survive, but like other forms of acute delirium it is generally fatal. When it occurs early in the disease, as it sometimes does, it may form the basis for a diagnosis of "galloping" paresis, some of the reported cases of which are probably examples of this condition. It may be said

here that rapid cases of paresis are not confined to this or to any agitated type, as the convulsive or apoplectic attacks may be fatal almost in the earliest stages of the disease.

Remissions of the symptoms may occur at any time prior to the appearance of complete dementia, and last in some cases for years. Sometimes these remissions are so complete as to suggest recovery, and cases have been reported as such. More often there is a check to the progress of the disease, which still manifests itself in some ways; the patient remains in a stationary condition, and one that in itself alone might not suggest paresis. The longest continuous remission of this kind personally observed by the writer was about five years, which is much above the average, though cases of much greater duration have been reported. After the third or final stage has appeared, remissions are hardly to be looked for.

The passage from the second to the third stage of paresis may be sudden or gradual. Generally, sooner or later, in the second stage, the mental failure becomes more marked and rapid, the paresis more pronounced. The patient becomes neglectful of the ordinary decencies with the advancing dementia, and the third or final stage may be said to have begun. The dementia and the paralysis, however, do not by any means progress *pari passu;* in some patients the dirty habits precede the physical symptoms, and vice versa. In the typical form this third stage is characterized by the aggravated speech defects, increased feebleness and inco-ordination, marked failure of mentality in all respects, as well as by the untidiness. Symptoms of cortical irritation are more frequent, the convulsive and congestive attacks become more frequent and also often more severe, with correspondingly more rapid advances in mental and physical deterioration, tremor becomes more marked, and such symptoms as grinding of the teeth, localized spasm, contractures, etc., are frequent, and the former may be almost constant, nutrition fails, trophic disorders such as bedsores, ulcerations, buttock sloughs, hæmatoma, arthropathies, fragility and fractures of bones, etc., make their appearance. Deglutition is embarrassed, and special care has to be taken to prevent choking from food. Any or all of these symptoms may coexist, and the final stage of all cases which are not carried off earlier by accidents incident to the condition or by intercurrent disease is a gradually failing vegetative existence, ending in death from final gradual exhaustion of nerve centres, with cardiac and respiratory failure or disease of lungs, kidneys, digestive tract, etc.

The average duration of a case of paresis is about three years from the date of first recognition of the patient's aberration, but it varies between very widely different extremes. The shortest case personally observed by the writer was of apparently less than two months' duration; the longest, possibly eight or ten years. The preliminary stage may extend over years, while long remissions or periods of non-progression may lengthen out any stage of the disorder before the final general physical breakdown.

Diagnosis.—In the fully developed stage of paresis there is little difficulty in the diagnosis, particularly if the characteristic speech alterations and the peculiar facies which becomes familiar to all who have to do with these cases are present. It is in the earlier stage, when the physical symptoms are less developed, that the difficulty exists. The disorders that are then likely to be confused with it are neurasthenia—itself a protean affection—simple mania, and circular insanity. It is usually said that the moral deterioration as well as the actual mental deterioration is sufficient to distinguish the early stages of paresis from neurasthenia, but this is a hardly reliable test for all cases. There are incipient cases of paresis in which the moral and æsthetic sensibilities are not so obviously impaired, and cases of neurasthenia in which there are actual defects of memory and conditions of mental incapacity that might lead to error. It is not always possible to distinguish beyond question in the earliest beginnings the symptoms of the irreparable changes in the nerve cells from those of the less serious disorder. There is usually, however, a less serious aspect to the depression symptoms and a more gradual development in neurasthenia. The prior history aids in the diagnosis; in paresis the symptoms appear in a more sudden manner and in all cases time soon reveals unmistakable distinctive characters in the earlier physical symptoms, the abnormal pupillary reactions, the tremor and changes in the facial musculature, and the usually graver nature of the mental defect. Simple melancholic depression may sometimes be simulated for a short time, but simple maniacal excitation or hypomania, or still more the excited stage of circular insanity, is liable to cause mistakes. The type of mental derangements in some cases of hypomania and circular insanity is very similar to that of the exalted phase often observed in incipient paresis, and the circular type of the latter is sometimes even still more confusing. I have in mind a noted case of circular insanity who has been pronounced a paretic several times by alienists of experience. Here also the element of time aids in the diagnosis; the circular cases do not show the progressive dementia or the physical symptoms as they do even in the circular form of paresis.

The organic brain disease of arterial sclerosis or atheroma is another condition that has, in the writer's opinion, given rise to more erroneous diagnoses of paresis than any other. The clinical symptoms may closely resemble those of paretic dementia, using the term in its strictest literal sense. The transient congestive or convulsive attacks, the gradually advancing mental failure, the motor symptoms, and even the peculiar speech defect may to some degree be imitated, though the latter is seldom a perfect reproduction of paretic speech. The pupillary reflexes are also less characteristic, and the age and history of the patient usually clear up the diagnosis. Many cases of paresis in individuals over sixty years of age, however, have been reported, and I am inclined to think that most of them were really cases of senile organic dementia. Occasionally a case of this kind takes on the galloping form of acute delirium, thus rendering the resemblance more striking, and in most cases, though not in all, the prognosis is not much better than in true paresis.

Various toxic agents, alcohol, lead, arsenic, and the toxin of pellagra, now and then produce syndromes like that of paresis. It is possible, as already said, that there may be other toxins than that of syphilis that act as predisposing or even as direct exciting causes in exceptional cases. Possibly those mentioned may act in this way; the terminal paralysis in some cases of chronic alcoholism has sometimes a strong suggestion of the demented type of paresis, though there are characteristic differences. A temporary condition very closely resembling certain phases of paresis caused by this agent is well known. In some cases of chronic lead poisoning there is a close resemblance to paresis. Regis classes this among the pseudo-general paralyses, and finds it often only temporary and curable. It has, he says, a more rapid onset and course, and the symptoms, however severe, are apt to disappear with the elimination of the poison. Personally I have observed at least one case of chronic lead poisoning with some of the features of paresis, which was diagnosed as such by a skilled alienist. The patient died in a convulsive attack. The macroscopic appearances of his brain were not unlike those of paretics, and there was also an interstitial nephritis. So far as the diagnosis between this and the usual form is of any importance, it can be made as a rule by the history and the usual symptoms of plumbism.

Very much has been written in regard to the diagnosis between syphilitic pseudo-paresis and true general paralysis. It is said that the former can be distinguished by the irregularity of its course, the character of the ocular muscle palsies, differences of speech disturbance, and character of the delusions; the sensory symptoms, headache, etc., are said to differ; optic neuritis is early and acute; and, lastly, it is claimed that the syphilitic disease yields to specific medication which is ineffective in paresis. These are only some of the differentiating points

alleged to exist between them, but each different writer brings out new arguments in favor of the distinction between the two. So far as personal experience goes, the writer agrees with Berkley that in the present state of our knowledge a differential diagnosis between syphilitic disease of the cortex and paresis is often impossible. When, after recent syphilis, we have as far as possible the complete syndrome of paresis, and as resistant to specific treatment as is paresis, it seems to me useless to venture a distinction between the two. It is not necessary or even reasonable to assume that the toxin of recent syphilis may not affect the cortical neurons under favoring conditions so as to produce the disease that the same toxin favors after a lapse of years. Our experience does not warrant a belief that the element of time is in any way essential. True paresis coexists sometimes with tertiary or even with secondary syphilis, and a gumma of the base may be found simultaneously with the paretic changes in the convexity. Of course there may be a diffuse, cortical, brain syphilis that is not paresis, but when the clinical picture of the latter exists and the condition consistently follows its course, there is no reason for calling it anything else.

The other disorders commonly mentioned as liable to be confused with paresis, such as multiple sclerosis, cerebral tumor, etc., require but brief notice. In multiple sclerosis we have the intention tremor, the quite different character of the speech defect and gait, and the lack of characteristic mental symptoms of paresis. It is very rarely that a brain tumor alone presents features that would lead to a diagnosis of paresis.

The importance of the diagnosis of this disease is chiefly in its earlier stages, especially in the earliest or prodromal stage. This is not so much for the possibility of successful treatment, which is very remote at the best, but on account of the need of control of the patient before he can do harm by his extravagances, and occasionally this has a forensic importance. The paretic, in the incipient stage, may seriously involve himself or friends and may impose upon others to their disadvantage. It is advisable that insurance examiners, especially, should be posted on its earliest symptoms and indications.

Prognosis.—The outlook for a paretic is bad in all respects. Sooner or later, and generally within a few years after the recognition of his disorder, he succumbs either to one of the complications incident to his condition or finally to the exhaustion or marasmus of the final stage. A paretic's life is far more uncertain at any stage than is that of victims of most other forms of insanity, and there is no time when a sudden and fatal aggravation may not occur. Nevertheless, there are now and then instances of a stay or remission in the disorder, and sometimes even a marked improvement, so that the patient is able to return again for a time to his former associations and occupations. Cases of cure have even been reported, some of them probably based on these remissions. The characteristic of the disease, however, is its fatal progressiveness, and all reported cures should, as Kraepelin remarks, be looked on with suspicion. While it may be possible that cures have occurred, they are so rare as to make the probability of one in any given case altogether a negligible quantity. The disease may be set down, in the present state of our knowledge and therapeutic resources, as inevitably fatal.

Pathological Anatomy and Pathology.—In cases of paresis in which an autopsy is afforded in the early stage of the disease by deaths occurring from convulsive or congestive attacks or other accidents, the chief macroscopic appearances are those of a chronic meningo-encephalitis often modified to some extent by the immediate conditions preceding death. Thus, for example, if the patient has succumbed very early from convulsions, or especially if an attack of acute delirium has preceded death, the gross appearances of the brain may be more or less those of an acute meningo-encephalitis with foci of intense congestion and hemorrhages. Ordinarily, however, the appearances are characteristic: there is marked congestion of the membranes and cortex, often localized more prominently in one hemisphere and generally most marked in the anterior and middle regions of the convexity; there are distention and tortuosity of the pial vessels, the membrane itself is thickened and opalescent or milky, and the cortical substance in certain regions, especially along the median fissure anteriorly, is apt to be adherent to the membranes and these to the cranium, so that in removing the calvarium it is sometimes almost impossible to avoid tearing away portions of the brain over the summits of the convolutions. This is almost sure to occur in separating the pia-arachnoid. The dura itself is sometimes largely adherent to the inner cranial surface, and the Pacchionian granulations are often excessively developed. There may be subdural hemorrhagic foci or cysts, especially in cases that have lasted for some time with symptoms of the developed disease, and in advanced cases there is always a lessening of the volume of the cerebrum with atrophy of the convolutions, and the adhesions of the membranes and cortex are less noticeable. The cranium itself may be thickened in these older cases. The ventricles are liable to be dilated and often granular on the ependyma. The white matter of the centrum ovale is congested, dotted with minute bleeding vessels, the thickened walls of which can sometimes be felt like bristles on passing the finger over the freshly cut surface.

The brain atrophy, according to Mott's observations, affects particularly the frontal and central regions and the hemispheres rather than the basal portions and the cerebellum. In juvenile paretics he found the left hemisphere weighing less than the right, a fact which he thinks has a bearing on the theory of overuse or stress as an exciting cause of the disease. In paretics generally the region of the brain most subject to stress is that most often atrophied. Œdema of the brain is another common and striking feature in post-mortems of paretics.

The microscopic alterations in this disorder involve the membranes and all the cortical tissues, the nerve cells, the neuroglia, and the vessels of the brain, as well as the nerve fibres of the brain and cord and even the peripheral nerves. Together with the lesions of pachymeningitis and the leptomeningeal framework of the pia-arachnoid with the various attendant vascular alterations, there are still more important changes in the cortical substance itself. There are marked vascular inflammatory changes which in the acuter forms may be most intense. In these cases of galloping paresis not only the cortical but the basal finer arteries in the pons and elsewhere reveal a very marked periarteritis, the coats are thickened while the lumen is not decreased but is clogged with granular matter and débris of blood, while externally the lymph spaces are prominent and there are collections of granular cells clinging to the vascular walls and minute extravasations throughout the nerve substance. To a certain degree these appearances are characteristic of all comparatively recent cases of paresis, though not so marked as in the hyperacute form. In the older cases we have a decided increase of the blood-vessels, with thickened walls and occasionally minute aneurysms, relics of old microscopic hemorrhages, patches of softening, etc. It is in the nerve cells themselves, however, that the most significant changes are met with, though these may not be so prominent in the earliest stages of the disorder. Kraepelin mentions as the earliest appearance a swelling of the nerve cell in which the nucleus may also be involved, but Berkley says he has never met with this in the paretic brain. In the acute processes, according to the former authority, there is a rapid cell degeneration (liquefaction?), while in the more usual slowly progressive cases the process is more a gradual sclerosis of the cell substance. The cell body itself shrivels up, the processes become tangled, the nucleus as well as the cytoplasm atrophies, and in certain regions there is almost a complete destruction of nerve cells in the later stages. The degenerative changes are, in fact, localized to a large extent, and the pathological appearances described may be apparently absent in other cortical tracts, at least until the later period when the morbid changes have involved the brain generally.

The nerve fibres also suffer with the nerve cells, and in the later stages of the disease the degeneration may involve the greater part of these in extensive cortical regions and be traced down to the basal ganglia and the medulla. A peripheral neuritis in paresis has long been observed, and the spinal implication has been studied particularly of late years, especially with reference to the relations with tabes. These relations were noticed or suspected more than twenty years ago by Türck, Horn, Spitzka, Kiernan, and others, and their recognition has recently become quite general, especially since statistical studies have made evident the common specific etiology of the two disorders. It is probable that, as Fuerstner says, there are few cases of paresis without implication of the cord, but the fact that it has not heretofore been the general custom to examine it as carefully as the brain has prevented its being so universally recognized. In the tabetic cases the lesions may be those characteristic of tabes or primary degeneration of the posterior root fibres and posterior column sclerosis, but it is probable that in the majority of cases a descending degeneration of the pyramidal tracts will be found more prominent.

The neuroglia is also involved, there is proliferation of the spider cells, the processes of which form a tangled mesh, and in some portions, especially the internal and external limiting layers of the cortex, this is particularly noticeable, the more so from the more or less complete disappearance of the normal nerve cells in these parts. With this sclerosed condition the cortex is sometimes reduced to half its normal thickness. Every portion of the nervous system is liable to be affected in this disease, the changes varying according to the situation and the function of the part. Secondarily to the nervous lesions we have the possibility of any and every other organ being affected, and some of the important viscera are as a rule more or less involved. The kidneys are very commonly found disordered, there may be gastric or intestinal lesions and cardiac or pulmonary complications are common.

The question whether paresis is primarily a disease of the nerve cells itself or of the vascular system has been extensively argued in the past, and at the present time the weight of opinion seems to favor the view that in this disease, as in tabes, the primary lesion is in the degeneration of the neuron, that it is primarily a parenchymatous rather than an interstitial disorder, with secondary inflammatory conditions of the vessels and tissues generally. Berkley, however, among recent writers favors the theory of the primary vascular disease, and Chalmers Watson has recently argued for the same in tabes. Either view, however, is consistent with the toxic origin of these disorders; it is perfectly supposable that in the more acute and rapid forms the vascular involvement may be simultaneous with or locally precede that of the nerve substance, while in other cases, especially those of the late developing type, the damage to the neurons may have been primary. Assuming that the toxin is that of syphilis, as is probable, we know that it may lie latent in the organism for long periods, to be finally wakened into action by various exciting causes, or it may act violently on the nervous system very soon after its introduction into the body. Virchow's dictum, quoted by Mott, "that a cell nourishes itself and is not nourished," is, as the latter says, probably the key to the pathology of this disease; but this does not exclude the possible earlier action of a toxin on the vascular system than on the nerve elements in certain cases. It is permissible, however, to suppose, according to the latest evidence, that in the majority of cases of paresis the neurons weakened by toxic action give way under conditions of stress, waste exceeds repair, and the early symptoms are those of neurasthenia. Under the influences of a present poison, however, brain waste goes further, the degenerative processes give rise to waste products in the cerebrospinal fluid and blood, and, these being themselves toxic, react on the neuron structures by irritating and causing conditions tending to inflammation and lymphatic and venous stasis, arterial anæmia, and œdema. The circulation is thus rendered unstable, the cortical irritability is increased, and we thus have, besides the action of the original toxin, a continuous vicious circle of waste, production of toxic products—cholin, nucleo-proteid (Mott and Haliburton), these again reacting on the nerve element through the lymphatics and the blood. The various symptoms of paretic dementia can all be accounted for by this continuous and progressive intoxication and auto-intoxication of the brain.

In tabes the process is a similar one, and the relations of the two disorders are coming to be generally recognized. Their differences are essentially due to the different portions of the nervous system primarily involved—in paresis the cerebral association system and in tabes the exogenous afferent spinal neurons, as pointed out by Mott. The selective action of the luetic toxin is exhibited in both, and, as already noted, they not infrequently appear to be combined or to follow one another in the same individual. In each disease the parts first involved appear to be those subjected to special strains—in paresis the cortical neurons from worry, alcoholic or other excesses, overwork, etc.; in tabes the spinal sensory neurons from similar causes affecting the cord, the strain of special overwork or equilibrium, sexual excess, exposure, etc. In each we have the specific pupillary reactions, the meningeal thickening, interstitial sclerosis, and wasting of the neurons. The pathological resemblances are certainly close enough to suggest a relation between the two disorders, and, taken in connection with the known facts of their etiology and course, the belief in their essential identity is certainly strongly favored, if not absolutely confirmed.

Treatment.—Inasmuch as paresis is essentially an incurable disease, the treatment is mainly palliative. It may be that, could it be taken in its very beginnings, there might be some hope of cure; but the opportunity is seldom if ever afforded. Still if a course of iodides is well borne it is worth trying, and the writer has sometimes seen what he thought was temporary improvement from it. He has had less experience with the mercurial treatment, but it is strongly recommended by Berkley. In some cases, and in advanced stages in all, specific treatment is ill borne and should not be carried far. Various general remedies have from time to time been recommended, but none have won much confidence. A judicious use of hydrotherapy, baths, prolonged and otherwise, adapted to the case, and carefully administered and watching the effect, has been useful, and in some cases has apparently produced long remissions of the disease. Baths are of course an essential adjunct to the treatment, and strict cleanliness must be secured, especially in the demented conditions. Serum therapy has, of course, been suggested, and very recently Bruce has reported experiments with the injection of serum from cases in remission. The pathological theory on which this treatment is based—that paresis is an intoxication from the bacilli of the colon group (Ford Robertson)—will require a great deal of demonstration before it can be accepted, but as a therapeutic suggestion it should be mentioned. In any case, on general principles, it is advisable to protect the patient as much as possible from any effects of intestinal intoxication. Tonics, heart stimulants, hypnotics, local remedies for bedsores, etc., all will come into play, in the various stages of the disease, to meet occurring conditions. Drugs usually fail to do much good in the acute delirious attacks, but sedatives should be tried with due precautions. In the paretic status epilepticus injections of chloral or hypodermic medication may be tried.

It would be well if all incipient paretics could be put under a modified restraint; often a rest cure would seem advisable. When the disease has fairly manifested itself, asylum treatment is necessary and should be continuous except during pronounced remissions. If during these the patient is able to be at home, he should still be under medical surveillance and advice. In the closing scenes of the disease, the most that can be done is to meet the symptoms as they arise and make the patient's last days as decent and comfortable as possible. It is important,

it should be said, to watch the patient's eating, and give only such food as can be taken safely without danger of choking. The condition of the bowels and bladder and the trophic alterations will all require careful attention.

Henry M. Bannister.

XIII. INSANITY DEPENDENT ON CEREBRAL DISEASES.—The term organic insanity or that from gross disease of the brain is usually confined in classifications of mental disorders to derangements associated with existing arteriosclerotic or syphilitic conditions or with actual hemiplegia or tumor symptoms. Taken in its broader sense it would, of course, include other forms not usually thus designated, such as paresis, most if not all epileptic mental disorders, and also senile insanity due to the pathologic changes of old age in the brain. Here, however, the term is employed in its usual signification, meaning by it the psychic disorders accompanying apoplexies, neoplasms, general or local cerebral arteriosclerosis, traumatisms and syphilitic diseases of the brain. The distinction is rather an arbitrary one in some respects, but it has its convenience in affording a place for special consideration of a number of mental syndromes having a direct etiologic connection with certain lesions, and moreover often modified in their psychic manifestations by the character and extent of these lesions.

The distinction between insanity from arterial disease of the brain and from certain types of senile mental decay is not a very clear one, especially since the former occurs as a rule in the down-hill period of life and is attended with other more or less similar conditions of general bodily decay. It may often properly be counted as a rather more pronounced form than usual of senile insanity, differing from the usual type only in its more marked symptoms and its commonly earlier appearance. "That a man is as old as his arteries" is a wise medical saw, but in most cases the brain wasting from rigid arteries does not cause insanity, and senile dementia when it appears comes on in the later stages of life and in less pronounced a form than is the form here under consideration. There are, as we understand it, two conditions of non-specific arterial disease that may cause general mental disorder, viz., atheroma, a well-known form of degeneration, usually senile, but occasionally premature, often more or less localized, and arteriosclerosis, a more general condition, usually toxic in origin, of arterial thickening and sometimes of atrophy in which the brain involvement may be secondary to or associated with like arterial disease elsewhere in the organism and especially in the kidneys. Binzwanger and Alzheimer have described a type of insanity associated with this condition, which may simulate the dementia form of general paresis with its paretic disorders of speech and frequent circumscribed paralyses and partial or complete temporary remissions. It is hard to say that this is a constant or even approximately frequent type from this cause, but a general demented condition may be said to be the rule in the advanced cases. There are, however, cases of marked arterial disease, more especially perhaps of the atheromatous type, in which the mental symptoms have a wide range, including both depressed and exalted states and conditions of chronic persecutory delusional insanity. With these, however, occur signs of gross cerebral disease such as convulsions, temporary hemiplegia, or spastic symptoms, etc. It is questionable whether we can safely allege that any one common type of clinical syndrome prevails, other than the dementia shown in loss of memory, irritability, mental confusion, lack of emotional control, etc., that are characteristic of these cases, and which are liable to complicate whatever special form of mental disease the condition may simulate. Perhaps we should add the pseudo-paresis due to this type of arterial disease, which is not uncommon and is, in the opinion of the writer, probably responsible for most if not all cases of alleged paresis occurring after the ages of fifty-five or sixty. The resemblance to the demented type of paresis is close in many respects, the occasional convulsive or congestive attacks, the paresis, etc., all closely resemble the true paretic dementia. There are generally, however, according to my observation, other symptoms of general diffuse arteriosclerosis which are lacking in true paresis, the speech is less characteristic, the disease less rapidly progressive, the remissions are rarer, and a specific history and the Argyll-Robertson pupil are lacking. There is a peculiar general facies in genuine beginning paresis—not meaning merely the facial expression, but peculiar to cases in their earlier stages; later, when the paralysis and dementia are more advanced, there is more resemblance between the two. The terminal stages are much alike so far as the clinical symptoms are concerned. The prognosis of this form is usually bad, but occasional temporary derangement of almost any form may occur in connection with the local minute aneurisms or hemorrhages of diffuse or local arterial disease of the brain.

Multiple cerebro-spinal sclerosis, with its typical symptoms of intention tremor, rigidity, nystagmus, etc., may also be attended with mental symptoms; a certain amount of mental dulness is probably the rule. It is not so frequent, however, to see what can be called actual insanity from this cause, and when it occurs it usually takes the dementia type, above described.

Hemorrhages of the brain are not necessarily dependent upon any diseased condition of the arteries and may occur at any age, indeed their greatest frequency is between early maturity and old age. The same is true of embolism, thrombosis, and tumors, all of which may be attended with symptoms of decided mental alienation. We can group for convenience' sake these derangements, the first three together, or perhaps rather the first two as post-apoplectic insanity, while the symptoms of thrombosis are liable to be slower in their onset and therefore not so properly designated as due to a stroke or sudden ictus. So far as the mental symptoms are concerned, however, there is no practical difference and the term paralytic insanity would apply alike to all three. It is probable that in every case of severe hemiplegia there is left at least a slight degree of mental impairment, but this is not always perceptible, even to rather close observations after the slighter attacks. It is only in the minority that we have what we can call actual insanity and this when it occurs may be but temporary. Sometimes, even after a minute hemorrhage, there may be a temporary hallucinatory delirium, but this is not accounted as true insanity. The most frequent change observed is a certain degree of mental incapacity, shown in inability to follow successfully former occupations, and a lack of emotional control. This may be temporary but often it is permanent, and constitutes a mild form of organic dementia that may not be sufficiently pronounced or serious to disqualify altogether the subject from social and business life. The younger the patient, the less likely is this condition to be lasting; in the older cases in which the atheromatous changes of senility have begun and progressed to greater or less extent, the mental weakness is apt to be progressive and a marked true organic dementia occurs. In the later stages these old hemiplegic dements are often helpless untidy paralytics: sometimes, however, the mental disorder takes on the form of ordinary terminal dementia or what is called usually chronic mania or melancholia according as motor excitement or depression prevails. Aphasic and other complications are frequent and post-hemorrhagic choreic symptoms are occasionally met with. The vegetative functions may be comparatively unimpaired, except in so far as they are embarrassed by the patient's lack of care of himself or by his paralysis. These chronic cases usually end in death from some intercurrent affection or a recurring paralytic stroke; sometimes from general breakdown with bedsores, and other trophic symptoms such as local gangrenes, etc. Kidney and heart diseases are, of course, not infrequent complications aiding in the fatal outcome. Convulsions are not rare and have also their influence or rather their evil significance.

Other forms of insanity may follow apoplectic attacks; probably the most frequent type is a confusional delirium

or mania, such as that already mentioned, but much more pronounced and serious and underlaid by a very decided general mental impairment. It may be called an agitated dementia with hallucinations and illusions, often permanent, though remissions and exacerbations may irregularly occur. In other cases melancholic symptoms may prevail with self-accusations and decided suicidal tendencies. These are sometimes well developed when the depression is not so apparent, but this is not usually the case. Delusional insanity with ideas of persecution is also sometimes observed after these brain lesions, and I have observed cases in which these symptoms were the only very noticeable after-effect of the paralytic attack, the patient appearing to all but the closest observers and to those of his own family as very nearly in his normal mental condition.

Thrombotic dementias, which are claimed by Berkley to be the most frequent of all, usually take on the form already described when speaking of arteriosclerotic disease of the brain, in which, in fact, they most commonly occur. There are more apt to be premonitions, headaches, vertigos, temporary aphasias, and local paralyses, drowsiness, etc., before the development of the hemiplegia which may itself be gradual in its onset. The mental symptoms may also be gradual in their appearance, or rather there may be precursory phenomena such as impaired emotional control, some loss of memory, etc., before the paralysis and the associated fully developed aberration. It sometimes, therefore, is slightly different in this respect from the other post-paralytic derangements, but there is no essential difference in cases from this cause and it may also occasionally give rise to diverse clinical symptoms of maniacal excitement, depression, delusions, etc. The subjects of thrombotic softening and its mental complications are apt to be of more advanced age than those of post-paralytic insanity from hemorrhage or embolism, unless, as is often the case, there is a specific taint combined with excesses and overstrain in its etiology.

The *pathology* of insanity from arterial diseases in the brain is necessarily complex and various in details. We may include it, however, under one general head, viz., disturbances of nutrition of the nerve elements, whether through irregularities of blood supply, from vascular rigidity and non-responsiveness to the normal vaso-motor regulation, or to direct injury or cutting off of the circulation in hemorrhage or arterial occlusion by emboli or thrombosis. In post-mortems we find the characteristic vascular lesions of arteriosclerosis or premature atheroma, and in long-continued cases brain wasting, especially of the frontal lobes, thickening of the pia, adhesions of the membranes, excess of Pacchionian granulations, sometimes pachymeningitis, cysts, organized clots, etc. The lesions of acute softening or of acute hemorrhage need not here be detailed.

The *prognosis* of arteriosclerotic mental disorder is not good, though that from hemorrhage or limited thrombosis is somewhat less serious. The liability to recurrences of apoplectic attacks or of extension of areas involved must be reckoned with and an apparent recovery of insanity following such accidents not be estimated with any too sanguine temper. The underlying condition has also to be considered, viz., cardiac disease, heredity, and any toxic agency that may be or have been active. Probably the least unfavorable prognosis can be given when the mental symptoms are due to a not too extensive softening from embolism in a person of otherwise sound cerebral organization. Here there may be no extensive arterial involvement.

The *treatment* is mainly symptomatic: quiet so far as it can be secured, attention to nutrition and elimination, securing of sleep, regulation of the circulation, etc. If there exists any bodily condition that aggravates the mental symptoms or threatens to do so it must be attended to.

Among other marked gross cerebral conditions that give rise to insanity, syphilis takes perhaps the foremost place. The amount of literature on this subject is very extensive, and the existence of a special recognizable type of mental disorder due to syphilis seems to be taken for granted by many alienists. There is no question but that syphilis is an antecedent or cause of very many cases of insanity other than paretics, who are generally coming to be considered as usually owing their disorder primarily to this cause. It is another thing, however, to admit that these cases are generally or even often of such a character as to form a clinical species of syphilitic insanity. Many of the best authorities deny this, while acknowledging that the close association of the specific disease and the mental disorder may be apparent. Syphilis has so many possible deteriorating effects on the organism that it can turn the scale easily between mental health and disease in those predisposed to insanity even before it manifests its special effects on the nerve centres. Its usual mode of attack on the brain is apparently through the vascular system or by the disturbing action of the neoplastic growths. The possible suspicion of lues in cases of cerebral arteriosclerosis has been already referred to, and the clinical symptoms of specific and non-specific arterial brain disease are not, in the writer's opinion, sufficiently different to permit their distinction so far as the psychic manifestations are concerned. The elaborately tabulated differential diagnostic points also between cerebral syphilis and paresis have not, so far as his experience goes, been found entirely reliable, and any differential diagnostic data between the mental symptoms of cerebral syphilis and those from non-specific arterial disease are still less constant. Paresis is a well-marked species of insanity, but in some of its phases and especially in its later stages it is often closely simulated by the dementia from arteriosclerotic or atheromatous disease. When recent or existing syphilis is associated with a clinical syndrome throughout like that of paresis there is no good reason for calling it anything else, and aside from such cases there are no really well-defined characteristic types of mental derangement that can be called syphilitic insanity—that is, so far as the strictly psychic symptoms are concerned. Of course, the association with the physical symptoms of cerebral syphilis, such as the paralysis of special cranial nerves, etc., makes the etiology evident and to a certain extent justifies the use of the term. Without these or a history of specific disease there would be very insufficient evidence of the existence of any such form. The most common type of derangement met with, apparently due to lues, is a gradually progressive dementia, but any other of the forms of insanity caused by toxins acting on the neurons or by arterial degeneration may appear. It is only in those possible cases of syphilitic insanity in which the poison overwhelms the nerve elements before it has acted long enough to produce its effect through arterial disease that it is likely we can find any really characteristic conditions, and our acquaintance with such cases is as yet too limited to enable us reliably to enumerate such differentiating characters. There is also a specific psychic factor present in some cases of syphilis which can hardly be called syphilitic in itself, but which may be prominent in the mental symptoms: that is, the moral effect of the existence of the disease. This, I think, I have seen to color, so to speak, the mental conditions in one or two cases in which the symptoms were of the depressive type and the organic disease certainly slight.

The mental symptoms of cerebral syphilis as described by authors are mainly a progressive dementia with sometimes a moral deterioration as a prominent feature in the earlier manifestations. Sometimes there may be a maniacal outbreak of brief duration or suspicious delusions. A special feature described by some is the changeableness of the symptoms. Some patients are for the most part quiet and apathetic, but occasionally have short spells of excitement and occasionally short spells of lucidity, again relapsing into the condition of lethargic depression. All the elaborate descriptions of the clinical phases of mental disease from syphilis, however, do not cover anything that cannot be shown to occur in insanity from non-specific tumors or arterial disease or from the action